we treated Y-27632 before acupuncture needling then assessed the nociceptive behaviors and mechanical threshold in the formalin and CFA induced mouse pain model.

Results: After acupuncture needling, ROCK2 was activated significantly 30 and 60 minutes later, whereas ROCK1 activation was not significant. Phospho-ERM was significantly activated 5 and 10 minutes after acupuncture needling. Acupuncture-induced ROCK2 and p-ERM expression were significantly attenuated by U0126, whereas, p-ERK and p-ERM expression was not attenuated by Y-27632. In the formalin and complete Freund adjuvant induced mouse pain model, acupuncture attenuated the nociceptive behaviors and the mechanical threshold. And these acupuncture analgesia was blocked by Y-27632 administration.

Conclusion: This study indicates that acupuncture-induced ROCK2 expression in the skin layer plays a trigger role in mediating acupuncture analgesia.

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http://dx.doi.org/10.1016/j.imr.2015.04.069

P1.063

Acupuncture induced local molecular signaling and its functional connectivity in the mouse brain

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Purpose: To identify the scientific mechanism of acupuncture therapy from peripheral to central, the molecular event at the acupuncture point and the neural activity of the brain regions after acupuncture needling were investigated.

Methods: Acupuncture was performed on GB34 acupuncture point of mice. After acupuncture needling, changes of proteins related to tissue deformation (Rho-kinase, ERM), neurotrophins (NT-3, BDNF, NGF), cell signaling (HSP27) and initiate immune (PRDX1, transketolase) were assessed. Then, the correlation between the molecular signaling was investigated. Next, to investigate the whole brain neural activity after acupuncture needling, c-Fos expression in thirty brain regions was investigated and partial least squares (PLS) analysis and network generation was performed.

Results: After acupuncture needling, Rho-kinase, ERM, NT-3 and HSP27 were up-regulated and BDNF, NGF, PRDX1 and transketolase were down-regulated in skin tissues at acupuncture needling point. Then we found that ERK activation worked as a trigger molecule to produce local molecular signaling. After acupuncture needling, c-Fos positive cells were significantly increased in the brain regions of cingulate cortex area 1 (Cg1), cingulate cortex area 2 (Cg2), primary somatosensory cortex (S1), secondary motor cortex (M2), insular cortex (Insul), piriform cortex (Pir), nucleus of solitary tract (NTS), dorsomedial periaqueductal gray (DMPAG) and lateral periaqueductal gray (LPAG) and decreased in the paraventricular thalamic nucleus posterior (PV) and the field CA1 of hippocampus (CA1). And these changes were inhibited by U0126 administration. Inter-regional correlations were significantly increased after acupuncture needling, and inhibited by U0126 administration. Among the brain regions, RMg, ST-DM, CA1 and NTS were determined as hub regions.

Conclusion: Acupuncture-induced ERK expression at acupuncture needling point plays a trigger role to acupuncture-induced cell signaling pathway, and also plays an important role in initiating central functional connectivity of acupuncture needling.

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http://dx.doi.org/10.1016/j.imr.2015.04.070

P1.065

Neuroprotective effects of the electroacupuncture at ST36 in trimethyltin-induced dementia animal model

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Purpose: In order to the neuroprotective effect of electroacupuncture (EA), the present study examined the effects of electroacupuncture in acupoint ST36 on trimethyltin chloride (TMT)-induced cognitive impairments rat using the Morris water maze (MWM) task and immunohistochemistry.

Methods: The rats were randomly divided into the following groups; naïve group (Normal), TMT injection group (Control), TMT injection and EA treated group in acupoint ST36 (ST36) and TMT injection and EA treated group in non-acupoint (Non-AC). Electroacupuncture (2 Hz, 2 mA, 10 minutes) was applied either to the acupuncture point ST36 the non- acupuncture point in the tail for the last 14 days. In the Morris water maze test, the animals were trained to find a platform in a fixed position during 4days and then received 60 sec probe trial on the 5th day following removal of platform from the pool.

Results: Rats with TMT injection showed impaired learning and memory of the tasks and treatment with EA in acupoint ST36 ($P < 0.05$) produced a significant improvement in escape latency to find the platform after 2nd day and retention trial in the MWM test. Consistent with behavioral data, treatment with EA in acupoint ST36 also significantly increased expression of choline acetyltransferase (ChAT) and acetylcholinesterase (AChE) immunoreactive neurons in the hippocampus compared to the Control group.

Conclusion: These results demonstrated that EA in acupoint ST36 has a protective effect against TMT-induced neuronal and cognitive impairments. The present study suggests that EA in acupoint ST36 might be useful in the treatment of TMT- induced learning and memory deficit.